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PAPER

TOXICOLOGY

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Postmortem Detection of Isopropanol in Ketoacidosis

ABSTRACT: Isopropanol (IPA) detected in deaths because of diabetic ketoacidosis (DKA) or alcoholic ketoacidosis (AKA) may cause concern for IPA poisoning. This study addressed this concern in a 15-year retrospective review of 260 deaths in which concentrations of acetone and IPA, as well as their ratios, were compared in DKA (175 cases), AKA (79 cases), and IPA intoxication (six cases). The results demonstrated the frequency of detecting IPA in ketoacidosis when there was no evidence of IPA ingestion. IPA was detectable in 77% of DKA cases with quantifiable concentrations averaging 15.1 ± 13.0 mg/dL; 52% of AKA cases with quantifiable concentrations averaging 18.5 ± 22.1 mg/dL; and in cases of IPA intoxication, averaging 326 ± 260 mg/dL. There was weak correlation of IPA production with postmortem interval in DKA only ($r = -0.48$). Although IPA concentrations were much higher with ingestion, potentially toxic concentrations were achievable in DKA without known ingestion.

KEYWORDS: forensic science, forensic pathology, toxicology, diabetes mellitus, alcoholism, ketoacidosis, acetone, isopropanol, bacterial conversion, alcohol dehydrogenase

Ketoacidosis can be observed in individuals with uncontrolled diabetes, excess alcohol intake, or starvation. While diabetic ketoacidosis (DKA) and alcoholic ketoacidosis (AKA) are well described, in some cases isopropanol (IPA) is detected in the blood of ketotic patients, which raises the possibility of concomitant ingestion of IPA (1). However, blood IPA in ketotic patients may also be due to the metabolism of acetone, a neutral ketone, to produce IPA by alcohol dehydrogenase (ADH, Fig. 1).

DKA is the most common form of ketoacidosis, responsible for 115,000 hospitalizations in 2003 (2) and 2459 deaths in 2002 (3). DKA is characterized by the triad of elevated blood glucose, acidosis, and the presence of ketone bodies in blood (4). Insulin deficiency, and the resultant production of counter-regulatory hormones (primarily glucagon), causes lipolysis and thus free fatty acid release from adipose tissues (5). These free fatty acids are subsequently oxidized in the liver to produce ketone bodies, which include acetoacetate, β-hydroxybutyrate (BHB), and acetone. Acetone is the least common of these metabolites, formed by the nonenzymatic decarboxylation of acetoacetate.

AKA is characterized by ketosis with an elevated anion gap and is associated with excessive ethanol ingestion and little nutritional intake (6). Free fatty acids are released from adipose tissues and metabolized in the liver to ketone bodies, as with DKA. Additionally, ethanol metabolism results in acetaldehyde production, which is oxidized to acetate, resulting in the conversion of NAD^+ to NADH and thus an increased NADH:NAD^+ ratio (7).

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ADH oxidizes alcohols to either aldehydes or ketones, with concomitant reduction of NAD^+ to NADH. In the case of IPA, oxidation yields acetone. There are seven recognized human variants of ADH, encoded by different genes on chromosome 4 (8). The presence of different variants of these ADH genes in individuals is associated with the varying risk of alcoholism and level of alcohol consumption in a population (8,9). However, ADH can also facilitate the reverse reaction, that is, the reduction of ketones to produce alcohols. If the ketone concentration is sufficiently high and the cofactor balance also favors the reverse reaction (i.e., a high NADH:NAD^+ ratio), then the reverse reaction can occur, for example, the production of IPA from acetone (10).

In clinical practice, IPA, or 2-propanol, is typically found only in cases of ingestion, and IPA intoxication is reported in roughly 7500 patients per year (11). IPA is a potent central nervous system depressant, with blood concentrations >50 mg/dL considered toxic. Concentrations over 150 mg/dL produce coma and hypotension (12,13), and a concentration of 340 mg/dL is considered lethal (14). However, there have been few reports in the clinical literature of IPA detection in patients in whom IPA ingestion was not suspected. Prevost et al. (6) described the case of one individual who presented twice with serum IPA in AKA in the absence of IPA ingestion. A case of DKA with concomitant detection of IPA has also been reported (15). Bailey (1) reported five cases of IPA detection in acetonemic patients in DKA, and Jenkins et al. (16) presented a brief summary of acetone and IPA concentrations in 162 postmortem cases. A few earlier reports also document finding IPA in ketoacidosis, whether diabetic or alcoholic (17–19).

In this article, we present a summary of 15 years of medical examiner cases where DKA or AKA was a primary or contributing cause of death. Blood concentrations of acetone and IPA are reported and compared with cases of known or highly suspected IPA ingestion. The purpose of this study was to demonstrate the

frequency of detecting IPA in ketoacidosis, the concentrations potentially encountered in both DKA and AKA, and any differences in IPA concentrations found in ketoacidosis compared with IPA ingestion.

Materials and Methods

Historical case review was conducted in deaths recorded by the King County Medical Examiner's Office, Seattle WA from 1995 through September 2010, for which an autopsy was performed and cause of death assigned by the medical examiner's office. Records were searched for those having laboratory results positive for either or both IPA or acetone. Decedents were classified into three categories: DKA, AKA, or IPA intoxication. DKA was assigned either if DKA was listed on the death certificate or if the decedent had a nonzero blood ketone concentration and diabetes was listed as a cause of death or contributing factor. Decedents were classified as AKA if AKA was listed on the death certificate or if the decedent had a nonzero blood ketone concentration and either chronic ethanolism/alcoholism or alcoholic fatty liver was listed as a cause of death. The diagnosis of AKA was therefore based on decedent history, scene evidence of ethanol abuse, and postmortem findings of alcoholic liver disease (cirrhosis or fatty liver), with no history or postmortem findings of diabetes mellitus. Postmortem testing for BHB, a more reliable marker for ketoacidosis (20), was not available for this study. Decedents were classified as IPA intoxication if IPA ingestion/intoxication was listed as a cause of death or contributing factor. Diagnoses of DKA or AKA were made based on accepted principles of forensic pathology, with DKA typically confirmed via urine dipstick analysis and blood toxicology and AKA commonly confirmed via history, autopsy findings, and toxicology.

Postmortem interval was calculated based on the estimated date of death, as recorded on the death certificate, to the date of autopsy. Data are presented as means plus or minus standard deviation. Statistical significance was assessed using one-way ANOVA with Bonferroni correction for multiple comparisons. Correlation coefficients were calculated in GraphPad Prism (GraphPad Software, Inc., La Jolla, CA) using a nonparametric (Spearman's) correlation calculation. Glucose measurements are not provided for diabetic cases as glucose is not a reliable marker in postmortem samples, because of the bacterial consumption (21). In some cases, an analyte (i.e., acetone or IPA) was detected but not quantifiable. When this occurred, the case was included in the analysis but excluded from the calculations of mean and standard deviation.

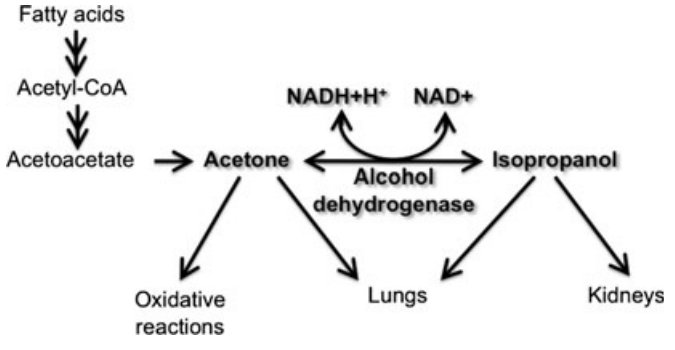

FIG. 1—*Isopropanol (IPA) production from acetone. During diabetic or alcoholic ketoacidosis, lipolyis and fatty acid oxidation produce acetyl-CoA, the substrate for ketogenesis and thus production of acetone. Under these conditions, along with a high NADH:NAD$^+$ ratio, alcohol dehydrogenase can produce IPA from acetone.*

TABLE 1—*Concentrations of acetone and IPA in DKA, AKA, and IPA intoxication.**

Condition	*N*	Acetone (mg/dL)	Ethanol (mg/dL)	*N*	Acetone (mg/dL)	Ethanol (mg/dL)	IPA (mg/dL)	Acetone:IPA†
	All Cases			Cases with IPA				
DKA	175	36.4 ± 21.2 [10,30,160]	11.5 ± 36.4 [0,0,220]	134	40.6 ± 23.3 [10,31,160]	14.2 ± 40.6 [0,0,220]	15.1 ± 13.0 [3,10,100]	3.42 ± 2.08
AKA	79	18.6 ± 12.5 [3,13,60]	59.8 ± 105.2 [0,0,450]	41	20.2 ± 15.4 [10,20,60]	71.8 ± 111.6 [0,20,450]	18.5 ± 22.1 [10,10,46]	1.90 ± 1.42
IPA intoxication	6	67.8 ± 51.6 [10,65,160]	92.5 ± 160 [0,0,160]	6	67.8 ± 51.6 [10,65,160]	92.5 ± 160 [0,0,160]	325.5 ± 260 [50,277,650]	0.27 ± 0.25

DKA, diabetic ketoacidosis; AKA, alcoholic ketoacidosis; IPA, isopropanol.
*Values are mean ± standard deviation; those in brackets are [minimum, median, maximum].
†Ratio of acetone concentration to IPA concentration.

Analytical measurements of ethanol, acetone, and IPA were performed by the Washington State Toxicology Laboratory using automated headspace-gas chromatographs (HS-GC; Agilent/Hewlett-Packard, Agilent Technologies, Santa Clara, CA) equipped with a flame ionization detector. Briefly, 0.2-mL aliquots from whole blood samples (harvested from either central or peripheral venous locations and preserved in sodium fluoride and potassium oxalate) were diluted, in duplicate, with 2.0 mL of internal standard (IS) (n-propanol + NaCl) in headspace vials. The vials were analyzed on two separate HS-GC instruments that differed only by the selectivity of their columns. Ethanol concentration in each sample was obtained from three-point calibration curve generated from aqueous calibrators run simultaneously with the samples. Two-point calibration curves were used to quantify acetone and IPA. Quantification was subsequently determined by comparing the peak-height ratio of volatile:IS to those obtained with the calibrators of known concentrations. The average of the duplicate results was reported. Results that were above the limit of detection (LOD) but below the limit of quantitation (LOQ) were reported qualitatively as positive. LOD and LOQ were 2.5 mg/dL and 10 mg/dL for both acetone and IPA, respectively. Although the LOD for ethanol was 1 mg/dL, only ethanol concentrations equal or above 20 mg/dL were reported. Concentrations equal or below 19 mg/dL were reported as negative.

The headspace operating conditions were as follows: vial equilibration with low shaking at 70°C (10 min), vial pressurization (0.17 min), sample loop fill at 85°C (0.15 min), loop equilibration (0.15 min), and transfer line temperature 125°C. Gas chromatograph operating conditions were as follows: J&W DB-BAC1 or DB-ALC2 capillary columns (Agilent Technologies) at 40°C and a helium carrier gas flow rate of 16.4 mL/min with inlet operating in split mode (1:1). A flame ionization detector was employed at 250°C with hydrogen and air flow rates of 40 and 300 mL/min, respectively.

Results

DKA was identified as the cause of death in 175 decedents, with a mean blood acetone concentration of 36.4 ± 21.2 mg/dL (Table 1). Approximately 75% of these cases also had IPA present in the blood, at an average quantifiable concentration of 15.1 ± 13.0 mg/dL. In cases with IPA detected, the average acetone concentration was 40.6 ± 23.3 mg/dL. The distribution of acetone and IPA concentrations is depicted in Fig. 2. As shown, IPA reached concentrations as high as 100 mg/dL, although most concentrations were below 50 mg/dL. Blood ethanol concentrations are given in Table 1.

AKA was identified in 79 cases, where the mean blood acetone concentration was 18.6 ± 12.5 mg/dL (Table 1). IPA was present in the blood of 52% of these cases, at an average concentration of 18.5 ± 22.1 mg/dL. Figure 2 presents the distribution of acetone and IPA concentrations in AKA cases.

Intoxication with IPA was recorded in six cases, where the average IPA concentration was 325.5 ± 260 mg/dL and the corresponding acetone concentration was 67.8 ± 51.6 mg/dL (Table 1). The concentrations of both IPA and acetone are significantly higher ($p < 0.05$) in IPA intoxication than in either DKA or AKA (Fig. 3). Calculation of the ratio of acetone to IPA concentration demonstrated a major difference between the cases of IPA toxicity and IPA arising from ketoacidosis. The average acetone:IPA ratio was 3.42 ± 2.08 in DKA and 1.90 ± 1.42 in AKA, compared with a ratio of 0.27 ± 0.25 in IPA intoxication. Although the ratio of acetone to IPA is substantially higher in both AKA and DKA, compared with IPA intoxication, the difference is significant only in the case of DKA ($p < 0.05$). Additionally, the acetone:IPA ratio in AKA is significantly lower than in DKA ($p < 0.05$).

Decedent demographics are provided in Table 2. In cases of DKA, 62% were men with an average age of 47. In cases of AKA, 71% of the cases in this study were men, with an average age of 51, while most AKA decedents were white (Table 2). We performed subgroup analyses of white and black decedents, under the hypothesis that there may be more similar expression of ADH isoforms within race groups, and therefore potential correlation between acetone and IPA concentrations. However, there was no significant correlation between acetone and IPA concentrations, either in the overall population or in white or black decedent subgroups.

Postmortem interval is the time between death and autopsy and was also included in the analysis. In Fig. 4, we present a dot-plot of the acetone:IPA ratio versus postmortem interval for DKA, AKA, and IPA ingestion. There is a slight negative correlation between the acetone:IPA ratio in DKA and postmortem interval ($r = -0.48$). This may be consistent with postmortem conversion of acetone to IPA by bacterial ADH (22). In the case of AKA and IPA ingestion, no correlation was detected, although smaller sample sizes limit the analysis.

Ethanol concentrations were also analyzed, and values are given in Table 1. We note that ethanol detection of <20 mg/dL was reported as negative. Substantial variability in ethanol concentrations was noted, while no significant correlations were revealed.

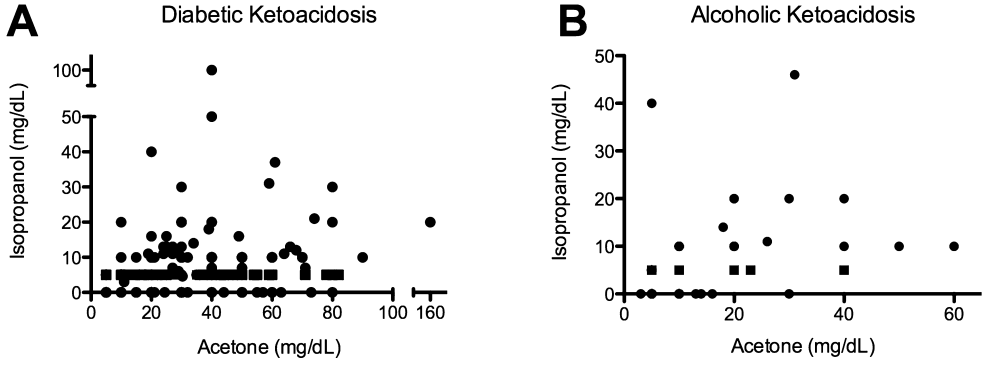

FIG. 2—*Scatter plots of isopropanol (IPA) and acetone levels in diabetic ketoacidosis (DKA) (A) and alcoholic ketoacidosis (AKA) (B) decedents. There is no detectable correlation between acetone and IPA levels in DKA or AKA. Square symbols represent cases with IPA levels that were detected but not quantifiable (arbitrarily plotted at 5 mg/dL).*

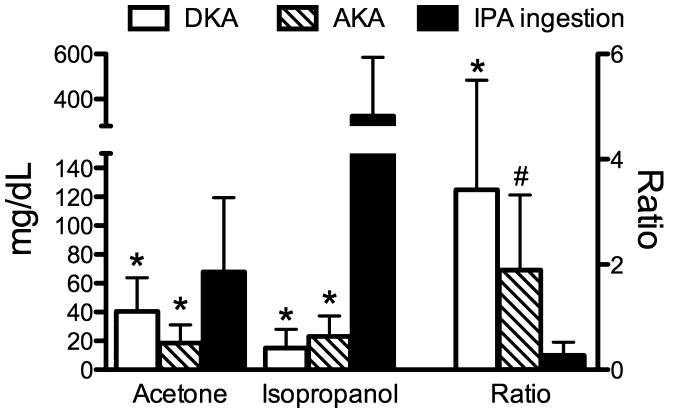

FIG. 3—*Acetone and isopropanol (IPA) levels in decedents with DKA, AKA, and IPA intoxication. Acetone and IPA levels are depicted on the left* Y*-axis, while the ratio of acetone to IPA is graphed on the right* Y*-axis. *indicates* $p < 0.05$ *compared to IPA intoxication and # represents* $p < 0.05$ *compared to DKA. DKA, diabetic ketoacidosis; AKA, alcoholic ketoacidosis.*

TABLE 2—*Decedent demographics according to condition.*

Condition	*N*	Age, Years*	Male (%)	Race W/B/O† (%)
DKA	175	46.7 ± 13.4	62.3	74/21/5
DKA + IPA	134	53.7 ± 18.3	61.2	72/24/4
AKA	79	50.3 ± 10.3	70.9	96/3/1
AKA + IPA	41	51.3 ± 9.0	79.5	93/5/2

DKA, diabetic ketoacidosis; AKA, alcoholic ketoacidosis; +IPA, cases with isopropanol detected.

*Mean ± standard deviation.

†Races: W = white, B = black, O = other (Asian/Pacific Islander or Native American).

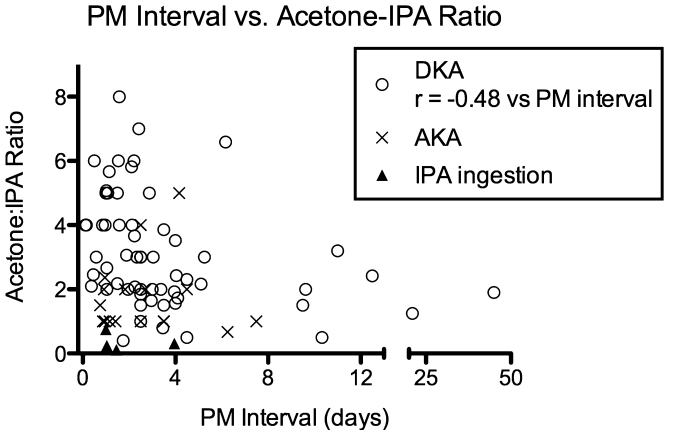

FIG. 4—*Postmortem (PM) interval analysis. PM interval is plotted against the acetone:IPA ratio for DKA, AKA, and IPA ingestion. In DKA, there is a slight negative correlation between the ratio and PM interval (r = −0.48). DKA, diabetic ketoacidosis; AKA, alcoholic ketoacidosis; IPA, isopropanol.*

Discussion

IPA detection is well recognized in death investigation and in cases of IPA ingestion, but is not typically encountered in other clinical scenarios. As a result, patients with ketoacidosis who also have IPA present in the bloodstream may be suspected of IPA ingestion. However, IPA can also occur in ketoacidosis without IPA ingestion. This study documents 260 cases in which IPA was detected in decedents examined over a period of 15 years by the King County Medical Examiner in Seattle, WA. In only six cases was there evidence of IPA ingestion; in 254 cases, the IPA was because of ketoacidosis, either diabetic (175 cases) or alcoholic (79 cases). In decedents with DKA as a cause or contributing condition of death, over 75% had detectable IPA in the blood. As common as it appears in this study, this phenomenon has been reported infrequently in the literature, either in clinical scenarios (1,6,15,18) or in postmortem analyses (23,24). In this study, we make the diagnosis of AKA based on the combination of decedent history, scene evidence, and postmortem findings. While the measurement of BHB is generally recognized as a more reliable marker for AKA (20), BHB testing was not available for this study.

A recent report by Molina (24) reviewed cases where IPA was detected on postmortem toxicologic analysis. Molina (24) found that diabetic decedents had median blood acetone concentration of 46 mg/dL and median IPA concentration of 11.5 mg/dL, while chronic ethanol users had median blood acetone concentration of 19 mg/dL and median IPA concentration of 15 mg/dL. The results of the present study are consistent with these earlier publications and further supplement the literature by comparing IPA concentrations in ketoacidosis with concentrations owing to IPA ingestion.

Overall, IPA may be present in DKA more often than appreciated in clinical medicine. The possibility that the presence of IPA correlates with increased mortality in DKA warrants further investigation. In ketoacidosis, the metabolism of acetone to IPA by ADH requires a buildup of both acetone and the reduced cofactor NADH (14). Therefore, it is reasonable to suspect that IPA is present only in the most severe cases of DKA, that is, when there are very high concentrations of acetone. The results of this study failed to support this possibility, however, as there was no significant correlation between the concentrations of acetone and IPA. Admittedly, it is hazardous to compare the results from decedents with those from living patients. For example, the elimination half-life of acetone (17–27 h) is much longer than for IPA (1–3 h) in living patients (25), but there are no similar data for decedents, and many reasons to suspect that metabolism is much different after death. Regarding the effect of concurrent ingestion of ethanol, there were no significant correlations between ethanol and acetone or IPA concentrations, despite a slight trend toward higher ethanol concentrations in both AKA and IPA intoxication.

There are many variants of ADH, and the specific alleles carried by an individual influence the kinetic properties of the resultant ADH enzymes as well as the risk for alcoholism (8). In isolation, there may be a correlation between acetone and IPA concentrations for a specific ADH isoform, indicative of an equilibrium between substrate and product. To evaluate the possibility of a genetic basis for differential IPA production from acetone, for example, because of different isoforms of ADH, comparisons were made with decedents separated according to race. However, we detected no correlation between acetone and IPA concentrations within white or black decedents. The absence of correlation between IPA and acetone concentrations probably represents multiple, uncontrollable variables in the decedent population beyond ADH isoforms, including metabolic state (e.g., $NADH:NAD^+$ ratio) and postmortem decomposition leading to the conversion of acetone to IPA. Therefore, the lack of significant correlations is not especially surprising. Overall, DKA is slightly more common in women as a hospital discharge diagnosis (26), although the death rate from hyperglycemic crisis is higher in men than in women (3). The present data are consistent with an increased risk of mortality from DKA in

men, although the relatively small size of our population (175 cases of DKA) precludes further analysis.

Postmortem interval correlates with a lower acetone:IPA ratio in DKA. Although this is a weak correlation, it may be consistent with postmortem conversion of acetone to IPA by bacterial enzymes. Within hours after death, bacteria from the normal intestinal flora begin to migrate throughout the body (27). Depending on the ambient temperature, bacterial production of ethanol can become significant, and bacterial activity also may contribute to the conversion of acetone to IPA (22). This study found that increased postmortem interval correlated with a decreased acetone:IPA ratio, which is consistent with postmortem bacterial conversion of acetone to IPA. Because of the uncertainty in time of death estimates, this result should be interpreted with caution. Molina (24) suggested additional postmortem causes of IPA production, including embalming or washing of the body with IPA before tissue procurement, but these suggestions are not relevant to the present study.

Endogenous production of IPA has been documented infrequently and the concentrations are fairly low (6,28). In the present study, IPA blood concentrations were typically low in cases of AKA and far less than the concentrations because of IPA ingestion. On the other hand, IPA concentrations in DKA ranged up to 100 mg/dL, which is potentially toxic (11). As the results in Table 1 indicate, the ratio of IPA to acetone may be of some value in distinguishing DKA and AKA from IPA intoxication. Nevertheless, it always remains necessary to correlate toxicology results with the scene investigation, decedent history, circumstances, and autopsy findings when assigning a diagnosis or cause of death. In summary, IPA is frequently detected in decedents with DKA or AKA, owing to its endogenous production from acetone by ADH. The presence of IPA does not necessarily indicate IPA ingestion, and the acetone:IPA ratio may be useful in excluding an exogenous source.

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